Primary Postpartum Hemorrhage

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SUMMARY

Postpartum hemorrhage is the outstanding cause of maternal mortality, and a redoubtable contributor to puerperal death from other causes, notably infection and renal failure. The clinical situations in which hemorrhage is liable to occur must be better known, so that anticipatory and preventive measures can be taken. Recent knowledge about defibrinated blood in women with degenerative changes at the placental site must be incorporated in the thinking and practice of physicians dealing with obstetrical cases. The indications, limitations, and hazards of the various anesthetic methods available for parturient women should be carefully considered in the circumstances of each case.

PRIMARY postpartum hemorrhage is excessive bleeding from the genital tract during the first 24 hours after birth. A blood loss in excess of 600 cc. is usually regarded as hemorrhage. The bleeding may occur before or after the third stage of labor is completed. Although the blood loss is usually external, in certain instances it may be more or less concealed. The bleeding may be sudden and furious, erratically repeated, or a slowly exsanguinating trickle. It may follow spontaneous or operative vaginal delivery, cesarean section, or cesarean-hysterectomy. Postpartum hemorrhage occurs in approximately 5 per cent of all deliveries. Puerperal hemorrhage after the first 24 hours is known as *late postpartum hemorrhage* and has been considered elsewhere.[6]

There are both immediate and delayed hazards in postpartum hemorrhage. The immediate threat is death from loss of blood and irreversible shock. Delayed complications are puerperal infection, transfusion reactions, renal failure from lower nephron nephrosis or cortical necrosis, thromboembolic disease, and finally, Simmonds' disease, or pituitary cachexia.

Most cases of postpartum hemorrhage can be anticipated, more can be prevented, and all can be treated. No woman should die of hemorrhage, the number one cause of maternal mortality, and a formidable contributor to puerperal death from other causes. The best treatment for postpartum hemorrhage is its anticipation and prevention. The several major causes of postpartum hemorrhage, although generally known, are here considered in the light of present knowledge:

Uterine Atony

Myometrial exhaustion, or what is more commonly called uterine atony, is undoubtedly the most frequent cause of postpartum hemorrhage. The usual predisposing factors are prolonged or precipitate labor, overdistention of the uterus, antepartum hemorrhage, deep anesthesia, and mismanagement of the third stage. Prolonged labor is usually owing to cephalopelvic disproportion, to uterine inertia, or to the premature and injudicious use of analgesics or conduction anesthesia. Cephalopelvic disproportion should be recognized in good time, and never permitted to eventuate in desultory labor. Primary inertia should be treated by support and stimulation, in contrast to secondary inertia which should be managed by rest and sedation. Labor can be prolonged many hours by starting continuous caudal analgesia prematurely; labor can be brought to a complete standstill for hours by the untimely administration of "terminal" spinal anesthesia. Postpartum bleeding is almost inevitable after the relaxation induced by deep anesthesia, particularly ether. Uterine atony follows precipitate labor just about as often as prolonged labor; and one should be cautious about the induction of labor, as induced labor is often prolonged or precipitate.

Overdistention of the uterus from multiple pregnancy, hydramnion, or excessively large fetus, is notoriously liable to be followed by postpartum hemorrhage. In such circumstances, the uterus should be decompressed slowly and general anesthesia used cautiously. Cross-matched blood should be in readiness.

Trauma of Delivery

Injury to the uterovaginal tract continues to be a major cause of postpartum hemorrhage. The anatomical lesions are cervical and vaginal lacerations, including rupture of the vagina, paravaginal hematomas, and rupture of the uterus. With most of these accidents the bleeding is external, in contrast to the more or less concealed hemorrhage which occurs with ruptured uterus and paravaginal hematomas. The external bleeding is typically a slow, steady trickle of bright (arterial) blood, in distinction to the darker blood which escapes from the placental site. The possibility of these complications should be thought of after precipitate delivery, after difficult obstetrical maneuvers, and with the parturition of elderly primiparae.

Laceration of the cervix is inevitable if delivery is attempted through an incompletely dilated os. Manual or digital completion of dilatation has no place in modern obstetrics; if conditions are propitious for vaginal delivery except for a persistent

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remaining ring of cervix, Dührssen's incision should be made. The first clue to a contracted midpelvis may be brisk bleeding from puncture wounds over prominent ischial spines. Lacerations of the vaginal vault are not uncommon after breech extraction, forceps rotations, and the inept use of Kielland forceps. A rotary or torsion force may actually rupture the vagina (colporrhexis) by creating a circular tear around the cervix, usually in the posterior fornix, which communicates with the peritoneal cavity. The bleeding with colporrhexis is usually furious because of laceration of the internal pudendal, middle hemorrhoidal, or vaginal artery. No woman should be returned to bed if there is a steady trickle of blood. The cervix and entire vaginal vault should be inspected if there is suspicion of trauma. Sometimes a physician has to rely on tactile sense to locate a laceration which is hidden by the invagination of its edges. Lacerations should be visualized (or palpated) and promptly repaired. A gently placed vaginal pack is often adequate for superficial abrasions, but if the vagina is packed too tightly bleeding may be aggravated by forcing the ends of the severed vessel apart; moreover, overpacking the vagina may cause neurogenic shock. In rupture of the vagina, if hemostasis cannot be obtained per vaginam, ligation of the hypogastric artery may be necessary.

Vulvovaginal and paravaginal hematomas are not uncommon and are usually not preventable. Backbleeding from a retracted submucosal vessel in the episiotomy or a laceration is the usual mechanism. By starting the repair two centimeters above the apex of the episiotomy or laceration, the likelihood of "missing" a submucosal vessel is lessened. Hematomas should be recognized early, evacuated, and bleeding points secured. If the process is left to nature, the extravasation of blood may extend past the broad ligament to the lumbar plate.

Traumatic rupture of the uterus occurs much more often than vital statistics would indicate. Deaths attributed to "obstetrical shock" are often due to unrecognized or disavowed rupture. Rupture should be thought of after every difficult forceps delivery, version and extraction, and breech extraction.[2] If there is any doubt about the integrity of the uterus, manual exploration should be done. If rupture is noted, the uterus should be packed, and a continuous transfusion given until arrangements are completed for laparotomy. Hysterectomy is necessary in about half of such cases, although in many instances the uterus can be saved by repairing the rent.

Errors of the Third Stage

More women die during the third stage of labor than during the first two stages combined. Proper management of the third stage is perhaps the obstetrician's greatest responsibility. The third stage is all too often the fatal climax of a neglected first stage and a traumatic second stage worsened by the effects of injudicious anesthesia. Full description of the proper management of the third stage cannot be undertaken here, but this much should be said: The conduct of the third stage should begin during the second stage by the deliberately slow delivery of the fetus to allow time for the uterus to accommodate itself to its diminished volume and thus peel off the placenta in a physiological manner. The Credé maneuver is dangerous and obsolete. No attempts to deliver the placenta should be made until it has separated, and the fundus is firmly retracted. Squeezing or kneading the uterus traumatizes the myometrium, dislodges thrombi from the placental site, and invites inversion. If and when deep anesthesia is used, it should be stopped the moment the fetus is delivered. Mismanagement of the third stage of labor was responsible for four out of seven cases of acute or subacute inversion recently reported from the Sloane Hospital for Women.[4] Vigorous bleeding at any time calls for immediate extraction of the placenta. In addition to massage, bimanual compression, and use of oxytocics, manual exploration should always be done if bleeding persists after the placenta is out, and it should be done before a tampon is placed. Retained placental tissue, a submucous myoma, rupture or inversion may be noted.

An emergency unit of type IV(0), Rh-negative blood should be kept refrigerated on every lying-in suite. The equipment necessary for laparotomy should be immediately available in or adjacent to the delivery pavilion. It is well to transfer postpartum women to a recovery room for the first 24 hours, so that they may be observed constantly by competent personnel. This would do much to prevent serious secondary relaxations, and the "floodings of the newly laid women."

Defibrinated Blood

Thanks mainly to the Boston group of obstetricians, more and more is being learned about the development during pregnancy of conditions making the blood incoagulable. Many physicians have observed cases in which the patient bled to death because the blood had lost the ability to clot. Hemorrhage not only from the placental site, but from surgical incisions, venipunctures, and even from the nasopharynx has been observed; and in such instances death may have occurred despite heroic measures, including ample blood replacement and hysterectomy. Since the report of Maloney and his associates[5] in 1949 a significant literature has accumulated on the subject of acquired afibrinogenemia of pregnancy. It is now known that whenever there is necrosis or degeneration at the placental site, there is a chance that particulate matter, thromboplastin, toxins, and anticoagulants can be absorbed into the maternal circulation. In extreme cases the woman's blood may become totally defibrinated and quite incoagulable. The hematologic findings include critically low values for fibrinogen and prothrombin, and often a circulating fibrinolysin is present. A sample of such venous blood when incubated will either not clot at all, or else the clot will be so fragile that the slightest vibration will cause it to disintegrate. When the plasma fibrinogen level is reduced from

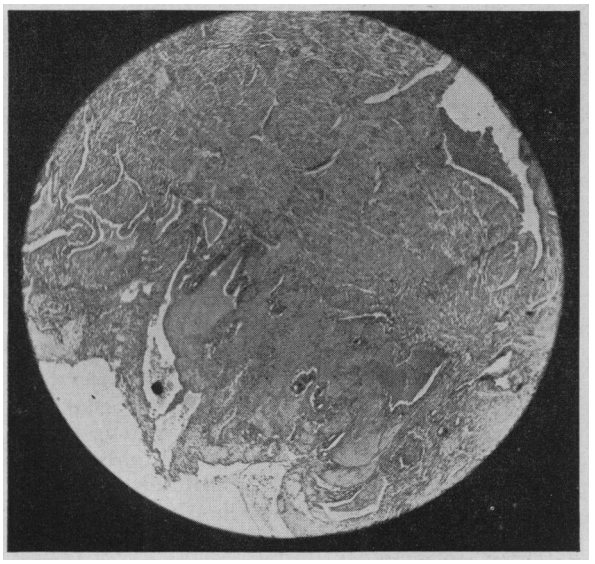

Figure 1.—Placental site six hours postpartum. Hysterectomy done for intractable hemorrhage due to afibrinogenemia. Note sinuses filled with liquid blood. × 17.

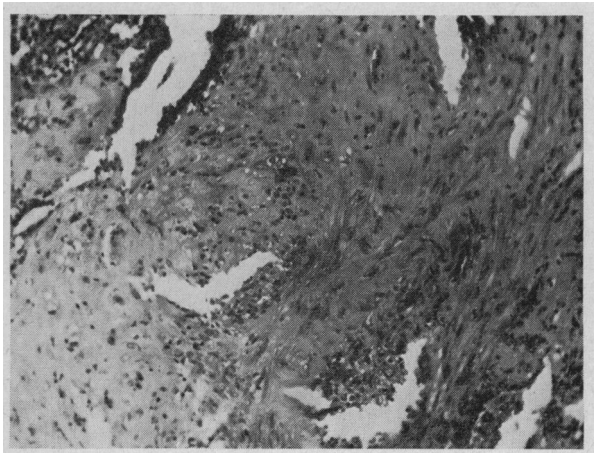

Figure 2.—Decidua basalis of same specimen as Figure 1. Note extensive necrosis of the decidual cells and round cell infiltration. The dead fetus had been carried in utero for four weeks prior to delivery. × 43.

the normal concentration of 0.5 gm. per 100 ml. to 0.2 gm. per 100 ml. the circulating blood is incoagulable.

It is now known that abruptio placentae, fetal death in utero, toxemia of pregnancy, and sometimes placenta praevia may be associated with defibrination of the maternal blood, depending upon the absorption of anticoagulants into the circulation. Women with these complications should be studied hematologically as well as obstetrically. In addition to replacement of whole blood, specific deficiencies of fibrinogen and prothrombin must be eliminated if uncontrollable postpartum hemorrhage is to be averted. Fibrinogen in the form of Fraction I, from which the virus of homologous serum jaundice has been removed by nitrogen mustard, is packaged in flasks of 2 gm. (2,000 mg.), and may be dissolved in 300 to 500 cc. of 5 per cent glucose or normal saline solution for intravenous administration.[3]

It is important that a uterine bag, or metreurynter, not be used in such cases. A uterine bag makes a closed system, so that when contractions begin, particulate matter, break-down products and anticoagulants are swept into the maternal sinuses, thence into the circulation, with risk of sudden death from amniotic fluid embolism, or aggravation of the incoagulable blood status. Simple rupture of the membranes, and the draining off of amniotic fluid with its noxious compounds, is a rational precautionary measure, and should be done when feasible.

In a recent case in which antepartum fetal death occurred at six months due to the Rh-factor, afibrinogenemia developed in the patient during the four weeks that the dead fetus was carried in utero. In examination of the specimen obtained at hysterectomy, which was six hours postpartum for intractable hemorrhage, unclotted blood in the placental sinuses was observed (Figure 1), and there were extensive degenerative changes in the trophoblastic-decidual junction (Figure 2), the source of the anticoagulants. Even though the placenta had been implanted high in the corpus the patient was admitted in early labor with prolapse of the placenta, no doubt due to the combined effects of necrosis of the placental site plus Braxton-Hicks contractions. In microscopic examination of the placenta (Figure 3) endarteritic obliteration of the villous vessels and atrophy of the chorionic epithelium were noted. Venous blood drawn at the time the patient was admitted to hospital did not clot even after five days.

Antepartum Hemorrhage

Antepartum bleeding predisposes to postpartum hemorrhage. The value of prophylactic blood transfusions in cases of placenta praevia and abruptio placentae was emphasized as long ago as 1919 by Bill[2] of Cleveland. The vicious circle of bleeding followed by uterine relaxation, followed by more

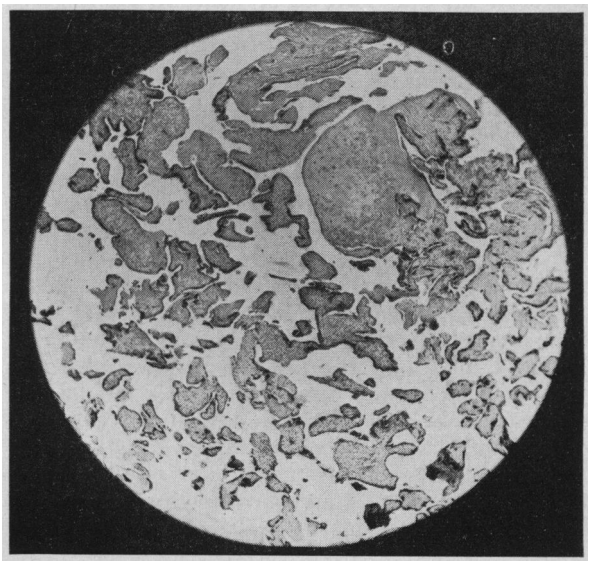

Figure 3.—Chorionic villi from same case as Figures 1 and 2. Placenta gradually became detached and eventually prolapsed. Note disappearance of chorionic vessels and epithelium. × 100.

bleeding until irreversible shock sets in, should be anticipated, and never permitted to occur. Blood lost antepartum should be replaced antepartum or intrapartum to prevent postpartum hemorrhage.

The interests of mother and fetus will best be served in cases of placenta praevia and abruptio by ample blood replacement, and simple rupture of the membranes, or cesarean section, depending upon the particular case. The author regards the use of dilating uterine bags as dangerous in either complication. In both conditions there is the risk of amniotic fluid embolism and of provoking the defibrinated blood syndrome. In placenta praevia there is the added hazard of furious postpartum hemorrhage from a laceration of the friable cervix or bleeding from a sinus in the placental site due to the trauma of a dilating bag. A bleeding sinus in the lower segment encountered at cesarean section can be visualized and transfixed. In both placenta praevia and abruptio placentae, hysterectomy is occasionally necessary as a life-saving measure.

Toxemias of Pregnancy

Postpartum hemorrhage should be anticipated in all cases of "toxic" pregnancy, for the following reasons: (1) Vasomotor collapse with consequent uterine atony and hemorrhage frequently follows delivery in such cases, for the vasomotor response to emptying the uterus is unpredictable. Therefore, spinal and general anesthesia should be avoided; local infiltration is preferable. (2) Often in "toxic" pregnancy there are degenerative changes at the trophoblastic-decidual junction, and the risk of defibrinated blood is considerable. (3) As was first pointed out by Slemons[8] in 1933, in toxemia of pregnancy the myometrium may be diffusely infiltrated with intermuscular hemorrhages identical with those observed in cases of abruptio placentae. A uterus thus affected may not retract postpartum, and hysterectomy may have to be done. In the case reported by Slemons, hysterectomy was necessary nine hours after cesarean section, because of repeated severe hemorrhages due to a blood-infiltrated myometrium.

Uterine Tumors

Uterine neoplasms are only rarely a cause of postpartum hemorrhage. Multiple intramural myomas or extensive adenomyosis[9] may interfere with proper retraction. More important, however, is a submucous myoma which has undergone red degeneration. Such a myoma can become agglutinated to the placenta, and prevent its complete separation in the third stage. If, upon inspection of the delivered placenta, a clean, punched-out defect is noted, a submucous myoma should be suspected and manual exploration carried out. Exceedingly rare causes of postpartum bleeding are cervical carcinoma, cervical polyps, ectopic decidua[7] which may involve the cervix, and hemangioma of the uterine wall.

Cesarean Section and Cesarean-Hysterectomy

Postpartum hemorrhage is a major cause of death following cesarean section. It is probable that the blood loss frequently amounts to between 500 and 1,000 cc. The sources of bleeding are the uterine incision, including transection of the uterine vessels on one side, unrecognized uterine rupture, and the placental site. If the placenta is attached to the defective scar from a previous cesarean section, the bleeding may be furious and intractable; Slemons[8] advised hysterectomy in such instances.

Hemorrhage should be anticipated in every cesarean section. There should be a functioning venoclysis with a No. 18 gauge needle started before the anesthetic is begun. In addition to preoperative blood replacement there should be a unit of crossmatched blood available in the operating room for even the "routine" case. If the operation is done for antepartum hemorrhage or toxemia, spinal anesthesia should be avoided because of the inherent risk of hypotension. Loss of blood will be reduced if the surgeon uses local infiltration. The placental site should be spared the trauma of rough sponging and the suction tip. Blood will be saved and morbidity reduced if myomectomy is avoided. In event of intractable bleeding from profound atony, placenta praevia, abruptio with uteroplacental apoplexy, rupture or perforation (from "failed forceps"), hysterectomy may be necessary.

The author has observed two cases in which the patient underwent cesarean-hysterectomy and several hours after operation lapsed into severe shock from internal hemorrhage. The increased vascularity of the pelvic structures in the pregnant state makes cesarean-hysterectomy a formidable undertaking. All vascular pedicles should be doubly ligated, as the risk of a major vessel's retracting is considerable. A progressively falling volume of erythrocytes as determined by hematocrit is the best laboratory indication of internal hemorrhage. If the physician is convinced there is continuing bleeding it is mandatory that he reopen the abdomen. Total hysterectomy is fraught with special hazards in the pregnant state, and, like all surgical operations during gravidity, should be avoided unless really necessary. Bleeding from the vaginal cuff and internal hemorrhage from vascular pedicles are calculated risks from this operation.

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Discussion by DONALD W. DECARLE, M.D., *San Francisco*

To me, there is but one obstetrical complication in the presence of which I must, at times, admit to actual panic. That complication is postpartum hemorrhage. This is particularly true in the presence of bleeding which one can not only see but also hear. I have known men far more experienced than I to be so completely unnerved in its presence that clear, sound judgment is lost. It is the complication which must always be feared as long as it is potentially present at every delivery.

As a subject for discussion, it has, therefore, been wisely chosen. It can never be exhausted as long as it leads the list of fatal complications in the field of obstetrics. As Dr. Melody has pointed out, in order to combat postpartum hemorrhage, one must be conversant with every phase of the conditions underlying it. This necessitates routine and frequent rechecking of the blood throughout every pregnancy. Potential or actual anemia as distinguished from the physiological hydremia of pregnancy can be recognized and properly treated before the onset of labor only by such a method. I deem this most important, since it can mean the margin of difference between life and death in the event of postpartum hemorrhage. The practice of placing the patient on iron therapy without such a routine hemoglobin evaluation only creates a false sense of security. It should be severely condemned.

For the most part, I agree wholeheartedly with Dr. Melody. However, in the matter of handling the third stage of labor, I still feel that the Credé maneuver, when properly carried out, is a valuable procedure. If one's hand is on the fundus during the third stage, certainly there is far less chance of the uterus' filling with blood concealed behind an undelivered placenta. The same is true of the Credé maneuver when an oxytocic agent is employed at the onset of the third stage. Otherwise, the placenta may become incarcerated.

Dr. Melody has very definitely changed our way of thinking on at least two points. In the first place, employment of the Voorhees bag has been largely replaced by more modern procedures. The danger of introduction of toxic material into the uterine sinuses by the use of the uterine bag should lead to complete and universal discarding of this procedure. Secondly, the same holds true of our attitude toward the fetus which has died in utero. It is apparently much safer to empty the uterus as soon as a diagnosis of intrauterine death has been made.

Through the efforts of the essayist, a supply of Fraction I is now available at Children's Hospital in San Francisco. It was my recent misfortune to watch as a patient died of bleeding which could not be controlled even by hysterectomy. Had this material been available at the time, such an outcome might have been prevented.